Results. There was a strong correlation between "non-invasively" measured cardiac output and traditional invasive technique, that remained over time. $r^2 = 0.90$ (implantation), $r^2 = 0.84$ (six and eleven months).

Conclusions. Repeated "non-invasive" measurements of cardiac output using an implantable monitoring system are feasible and has a potential for management of patients with severe heart failure.

960-62 Quinapril, Initiated After Advanced Ventricular Dilation in Patients With Chronic Infarction, Prevents Progressive Remodeling

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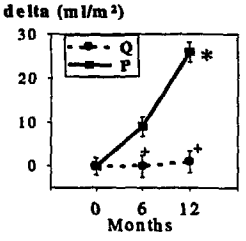
Patients with advanced progressive but not yet symptomatic LV dilation due to remote MI are at increased risk for adverse events. We tested the hypothesis that progressive LV dilation in chronic MI can be modified by the angiotensin converting enzyme inhibitor quinapril (Q) even if started years after MI. Of 138 patients with an MI 1.5 to 5 years before, 25 showed progressive LV dilation and were randomized (prospective, double-blind study) to placebo (P) or Q (10 to 40 mg/day). At baseline (mean ± sem), NYHA class was 1.2 ± 0.2 and 1.8 ± 0.2, age of MI 56 ± 6 and 55 ± 5 months, LV radionuclide ejection fraction (EF) 35 ± 3 and 39 ± 3%, end-diastolic volume (gated SPECT) 104 ± 9 and 117 ± 12 ml/m², wedge pressure (Swan-Ganz catheter) 10 ± 3 and 8 ± 3 mmHg in P (n = 13) and Q (n = 12), respectively. Progressive LV dilation continued in patients on P, but not on Q (figure; *p < 0.05 vs 0 months, +p < 0.05 vs P) while wedge pressure at rest (P: 12 ± 3, Q:9 ± 2 mmHg) did not change. Wedge pressure (mmHg) during bicycle exercise (25 Watt) was similar at baseline (P:20 ± 3, Q:20 ± 6), but at 12 months lower with Q (17 ± 4, p < 0.05) compared to P (24 ± 4).



Thus, further progression of LV dilation in chronic MI can be prevented by quinapril even if started years after MI. This may at least in part be due to prevention of exercise-induced rise in LV filling pressure.

960-63 Patients After Small Myocardial Infarction Are Not at Low Risk for Late Ventricular Dilation and Dysfunction

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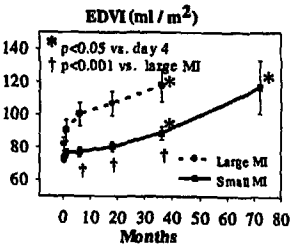
Left ventricular dilation after myocardial infarction (MI) is an independent predictor of adverse events and mortality. Asymptomatic patients with small MI are considered to be at low risk for LV dilation and late dysfunction and angiotensin converting enzyme (ACE) inhibitors are not recommended early after MI. To test this, we prospectively followed 104 asymptomatic patients after first MI for 7 years. Exclusions were ischemia, reinfarction and uncontrolled hypertension. At baseline, MI size (cineanglography) was 7 ± 1% (mean ± sem) and 30 ± 2% and radionuclide ejection fraction (EF) 53 ± 2% and 30 ± 2% in small (n = 87) and large (n = 27) MI, respectively. From 4 days, 4 weeks, 0.5 years, 1.5 years, 3 years until 7 years after MI, LV end-diastolic volume index (EDVI), by gated SPECT, shows a biphasic (fast-slow) in large and a monophasic (slow) kinetic in small MI (figure). This slow LV dilation in small MI was, however, followed by late deterioration of EF (4 days: 53 ± 2, 4 weeks: 54 ± 2, 1.5 years: 53 ± 2, 3 years: 49 ± 2, 7 years: 34 ± 3†, † ANOVA p < 0.05 vs. 4 days).



Thus, even after small MI, the LV may undergo slow but long-term remodeling which may be missed due to the flat slope of LV volume. Since it may precede insidious development of LV failure, even patients with small MI should be followed carefully and reconsidered for an ACE inhibitor.

960-64 Effect of Aspirin on the Acute and Chronic Hemodynamic Response to Angiotensin II Receptor Blockade With Losartan in Heart Failure

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Attentuation of the acute hemodynamic effects of ACE inhibitors by aspirin (A) in patients with heart failure (F) has been reported. Losartan (L), a direct angiotensin II receptor antagonist results in a favorable hemodynamic effect in F, but the interaction with A is unknown. We reviewed the data from our multicenter trial of 134 patients with ejection fraction < 40% and wedge pressure (PCWP) > 13 mmHg who had invasive hemodynamic assessments after the first dose of L and after 12 weeks of L (2.5, 10, 25, 50 mg). Aspirin was taken by 52% in both the acute and chronic study. A high dose L subgroup (25 & 50 mg, n = 51) was also reviewed. *Results:* For the entire population, the 5 hour acute and chronic hemodynamics were not attenuated by A for heart rate (HR), mean arterial pressure (MAP), mean pulmonary pressure (PAP), or systemic vascular resistance (SVR); p = NS by ANCOVA. Results of high dose group (25 & 50 mg L) is shown as change from baseline (+STD).

	Acute		Chronic	
	A(n = 22)	No-A (n = 29)	A(n = 20)	No-A(n = 23)
MAP	−4.8 ± 8.0	−7.2 ± 6.4	−3.1+6.3	−6.7 ± 7.2
PAP	−3.8 ± 3.4	−3.7 ± 4.5	−2.3+9.0	−6.9 ± 7.2
PCWP	−4.0 ± 3.7	−3.7 ± 4.7	−3.6+8.7	−4.1 ± 5.7

Conclusions: In this study population, A does not appear to result in a consistent attenuation of the hemodynamic effect of L in patients with F after both acute and chronic treatment.

960-65 Is Monotherapy With ACE Inhibition Preferable to Diuretics in Mild Heart Failure? A Comparison of Benazepril and Hydrochlorothiazide

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Theoretically, in mild heart failure without overt fluid retention ACE inhibition may be preferable to diuretics as monotherapy. As no comparative data exist, benazepril(B, n = 192) and hydrochlorothiazide (H, n = 195) patients (pts) with early, mild heart failure (NYHA cl II) due to ischemic or idiopathic dilated cardiomyopathy were compared during a 3-month double-blind, parallel study. After withdrawal of diuretics, if present, and > 2 days placebo, pts were randomised to 2 mg B or 12.5 mg H daily, with uptitration to 10 mg and 50 mg daily, resp., during the first month, and addition of the alternative treatment thereafter if necessary. Efficacy endpoints included bicycle exercise duration, NYHA class rating, discontinuation (worsening heart failure) and LV end diastolic dimension and fractional shortening (echo), assessed at 4, 8 and 12 weeks. Baseline criteria were comparable. In each group, 1/3 had never received diuretics and 1/3 continued digitalis glycosides (unchanged doses) for tachyarrhythmia control. 49% of pts remained on first-line medication alone, 50 B and 55 H pts requiring the highest dose. 33 B and 29 H pts discontinued due to worsening of failure or cardiac death, 8 and 6 and 2 and 1 B and H pts, resp. Exercise duration increased similarly, from 423 to 491 sec (B) and from 414 to 491 sec (H). 42% and 43% of B and H pts improved one NYHA class. Few pts deteriorated. Body weight did not change in either group. Neither drug significantly affected LV dimensions or function. Overall, side effects were comparable. However, hypokalemia (< 3.4 mmol/l) and worsening of renal function (increase creatinine > 30%) occurred in 15 and 14 H vs 2 and 8 B pts, resp. Whereas efficacy and general tolerability of monotherapy with B and H are comparable, electrolyte and renal function abnormalities prevail in H, which, in addition to neurohormonal stimulating properties of diuretics, favours ACE inhibitor monotherapy (benazepril) in mild heart failure without overt fluid retention.

TUESDAY POSTER